only seconds but recur with a variable frequency, usually in clusters and lasting from days to months. There are often spontaneous remissions for weeks, months or years between flurries of attacks.

About 70% of patients will obtain relief with the use of either carbamazepine or phenytoin. Thus, in about 30% of patients, medication is either ineffective or produces serious side effects necessitating discontinuance. These patients, therefore, may be candidates for the various surgical procedures that have been designed to treat tic douloureux. The mainstays of surgical therapy for this disorder are microvascular decompression of the trigeminal nerve via posterior fossa craniectomy, or percutaneous radiofrequency trigeminal gangliolysis. The former procedure has the potential of producing long-lasting pain relief without facial sensory loss, while the latter procedure avoids the risks of craniectomy and can be repeated easily if tic pain recurs.

Recently, retrogasserian injection of glycerol through a spinal needle inserted percutaneously into the trigeminal cistern has been shown to be an additional effective method of treating tic douloureux. The procedure is done with the patient awake under local anesthesia, and it is generally well tolerated. Pain relief from this procedure does not depend on producing sensory loss and there seems to be less likelihood of troublesome facial dysesthesias. Rare complications include corneal anesthesia and keratitis. More than two thirds of patients can expect pain relief for six years or longer and the procedure can be easily repeated in the event of pain recurrence. Experimental studies seem to indicate that the therapeutic effectiveness of glycerol in the treatment of tic douloureux is based on its nonspecific neurolytic effect, which may interfere with abnormal impulse generation within the nerve. Glycerol gangliolysis may represent a major advance in the treatment of tic douloureux because it is easily done, well tolerated by patients and produces much less facial numbness than other available neurodestructive procedures.

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Migrainous Stroke—New Therapeutic Considerations

MIGRAINE IS A common disorder, present in about 5% to 10% of the general population. The clinical spectrum of migraine varies widely, from patients with infrequent mild head pains to the unfortunate group with incapacitating pain. When the neurologic features of a migrainous episode, classically thought to result from vasospasm, dominate the clinical presentation, the term "complicated migraine" is used. In some patients with complicated migraine, the vasospasm is apparently so severe or prolonged, or both, that brain tissue in the distribution of the involved vessel suffers irreversible ischemic damage, evident clinically as stroke. Aggressive migraine prophylaxis is obviously indicated in this relatively small population of patients.

Several recent clinical studies have indicated that treatment with a calcium channel blocker (specifically, verapamil) may be useful in migraine prophylaxis for patients in whom more conventional agents fail. This class of agents may prevent migraine and, in particular, its vasospastic complications, both by inhibiting vascular smooth muscle contraction and by direct antiplatelet effects. Such actions would be especially desirable for patients at risk for migrainous stroke. Propranolol hydrocholoride, however, a drug widely used for migraine prophylaxis, may actually precipitate or prolong vasospasm through potentiation of α-adrenergic agonist vasoconstriction.

In patients considered at risk for migrainous stroke—specifically, those in whom the neurologic accompaniment is pronounced or persistent—ergot therapy is to be avoided. Propranolol may also prove unsuitable. Verapamil, in a dose of 80 mg three or four times a day, should be strongly considered for these patients. Other calcium channel blockers such as nifedipine may possibly be more effective, but verification is lacking at this time.

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When to Stop Anticonvulsant Therapy

BECAUSE OF potential side effects from long-term therapy, anticonvulsant therapy should be discontinued in patients with epilepsy as soon as there is a reasonable certainty that seizures will not recur. In practice, defining the point at which this reasonable certainty exists is a difficult question. More information is available for children than for adults. Studies of large numbers of children, with long follow-up, consistently show that about a quarter of patients who are seizure-free for four years while receiving anticonvulsant medication will relapse when the medication is withdrawn. Adults probably have a greater chance of relapse, though the precise relapse rate is not known.

There are clinical and electroencephalographic features that can identify patients who have a greater than average probability of relapse. Patients whose epilepsy took more than five years to control and those with intellectual impairment or other static neurologic abnormalities have relapses more than half the time. The presence of focal or mixed seizure disorders or the occurrence of unequivocally epileptiform abnormalities on an electroencephalogram predict a high relapse rate.

Certain forms of epilepsy have consistent clinical and electrographic features that serve to characterize them as benign epileptic "syndromes," including typical absence seizures with 3 cps spike-and-wave complexes that occur in children of normal intelligence. Similarly, children with focal motor epilepsy who have midtemporal and central spikes (the so-called benign focal epilepsy of childhood or Rolandic epi-

lepsy) should be considered to have a much lower than average relapse rate. Patients with these epileptic syndromes whose seizures have been controlled for four years should be considered for withdrawal of anticonvulsant therapy.

Certain potentially reversible illnesses may include recurrent seizures as part of their course. For example, metabolic derangements such as nonketotic hyperglycemia and profound hyponatremia may lead to frequent seizures that are difficult to control. Patients with alcoholism may have withdrawal seizures. None of these patients would be considered to have epilepsy, and anticonvulsant medication should be withdrawn when the underlying illness has resolved.

It appears reasonable to attempt withdrawing anticonvulsant therapy from patients, particularly children, whose epilepsy was easily controlled, who have been seizure-free for four years, who have at least average intelligence and normal neurologic function and whose electroencephalogram shows no abnormalities. This approach can be expected to yield the lowest incidence of relapse following withdrawal of medication.

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Neurologic Manifestations of AIDS

THE ACQUIRED immunodeficiency syndrome (AIDS) may be complicated by neurologic dysfunction in 30% to 40% of cases. Neuropathologic abnormalities, however, are found in about 70%; this may reflect the fact that subtle neurologic signs and symptoms may be overlooked in patients with severe systemic illnesses. In about 10% of patients with AIDS, the neurologic syndrome is the initial clinical manifestation. In such cases, herpes zoster radiculitis and aseptic meningitis are the most common harbingers.

Paralleling the nonneurologic effects of AIDS, the neurologic manifestations are most often due to opportunistic infections or malignancy. Meningoencephalitis, which may be acute or chronic, is the most common neurologic syndrome. In nearly 100 reported cases, most with brain abscess formation, *Toxoplasma gondii* has been implicated. About 60 cases of probable viral encephalitis have been reported, and although the specific virus has not always been discerned, both cytomegalovirus and herpes simplex virus have been cultured from the brains of such patients. *Cryptococcus neoformans* meningoencephalitis is third in frequency, with 33 cases reported. Less frequently implicated are other fungi, mycobacteria and spirochetes. Progressive multifocal leukoencephalopathy has also been reported.

Neoplastic involvement of the nervous system has been reported in 26 instances, including cases of primary brain lymphoma, metastatic lymphoma, meningeal lymphomatosis, epidural spinal cord compression and metastatic Kaposi's sarcoma.

Cerebrovascular complications, associated with nonbacterial thrombotic endocarditis, neoplasm and thrombocytopenia, have been reported in nine cases.

Peripheral nervous system complications occur less often (51 cases). These include chronic inflammatory polyneuropathy, distal symmetric neuropathy, cranial neuropathies due to lymphoma or inflammation, herpes zoster radiculitis and polymyositis.

Algorithms have been published for evaluating AIDS cases in which there are nervous system complications. Several caveats are noteworthy: first, toxoplasmosis is not ruled out by a negative computed tomographic scan or by "nondiagnostic" titers. Second, a cryptococcal antigen test may be positive in acellular spinal fluid. Third, herpes simplex encephalitis may be atypical in course and anatomic distribution in patients with AIDS. Last, different brain abscesses in the same patient may be due to different organisms.

Treatment is outlined by Snider and co-workers and Levy and colleagues. Frequent infectious recurrences, especially with *Toxoplasma* and *Cryptococcus*, raise questions about the duration of therapy.

Unfortunately, the overall prognosis is very poor in a patient with AIDS, with or without nervous system dysfunction. However, the nervous system complications themselves are frequently rapidly fatal if not diagnosed and treated; thus, such treatment may improve both quality and quantity of life in a patient who has AIDS.

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Medical Ethical Issues

MODERN RESUSCITATIVE TECHNIQUES, including ventilator support, often produce ethical dilemmas in treatment decisions for patients with two types of severe brain damage: brain death, in which the patient shows a complete loss of all brain functions, both cerebral hemisphere and brain stem; and states of permanent unconsciousness. One example of the latter is a persistent vegetative state in which deep coma evolves over a few days or several weeks into a condition of eyes-open unresponsiveness and sleep-wake cycles. Physicians and patients' families are increasingly questioning the indeterminate use of life-sustaining techniques for patients who are brain dead or who are extremely unlikely to return to cognitive, sapient life. The first group of patients is, by definition, medically and (but for a pronouncement) legally dead in most American states; the second group is very much alive and must be recognized as such.

The 1981 *President's Commission Criteria,* now the nationally accepted and most current standard for the clinical determination of brain death, superseded the 1968 *Harvard Criteria.* Neurologists (or, when appropriate, neurosur-